Cardiac Monitoring in a Community Hospital

Analysis of 18 Months' Experience

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■ *Cardiac monitoring facilities have been present in teaching hospital centers for over five years. A substantial decrease in mortality has been observed in monitored patients with acute myocardial infarction. The community hospital system offers a challenge to effective monitoring since many physicians care for patients and often many kinds of therapy are used.*

After 18 months of operation mortality from myocardial infarction was only 16.6 percent in a community hospital monitoring unit where the majority of the emergency care and resuscitation was carried out by nurses. Vital to this success was the use of standing orders for nurses, requirement of privilege to practice within the monitoring facility and acceptance of the nurse as a therapist in emergency situations.

Fourteen patients were successfully resuscitated and were later discharged from the hospital. Four of them had ventricular fibrillation from digitalis intoxication.

Patients with shock and severe congestive heart failure continue to be a major unsolved clinical problem. The results indicate that the potentially viable patient with serious electrical disturbances can almost invariably be salvaged.

THREE RECENT DEVELOPMENTS have been responsible for the institution of electronic monitoring facilities for patients with acute myocardial infarction. First is the availability of competent means of closed-chest massage and artificial ventilation. The second is the development and miniaturization of equipment capable of monitoring the electrocardiogram and other physiologic variables. Last is the realization that electrical rhythm disturbances constitute a major hazard to the patient with acute myocardial infarction. Many centers

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throughout this and other countries have initiated such units and have published the results of their experiences. [2,3,5-8,10,11,12,13]

Organization

In March of 1966 a monitoring facility was begun at Sutter Memorial Hospital, a 271-bed hospital without intern or resident staff. Planning and organization were directed by a committee of practicing physicians. Because of space limitations, four private rooms on a medical-surgical ward were equipped with conduits and wired for monitoring beds. Four more rooms were added later. The private room concept permitted solitude for the patient, yet rooms were within 30 feet of the nurses' station and access to resuscitation. Each room contained a cardiotachometer* with manual rate alarm setting, a line current-driven internal-external pacemaker, and a 5-inch oscilloscope mounted on wall brackets. No audible signal was present in the patient's room. A telephone jack in the wall bracket allowed a plug insert for synchronized DC shock. Inputs from the patient entered a wall faceplate, and conduit was run from this point to the monitoring equipment. Individual rooms were revamped to provide overhead lighting and eight major electrical outlets. Excess furniture was removed and suction equipment and manometers were added. An intercom system connected these rooms with the main nursing station and with the monitoring station. A completely equipped emergency cart with battery pacemaker, DC defibrillator, suction, bedboard, manual breathing bag, cardiac medications and other necessary equipment was immediately available in the hall.

A separate nursing station was constructed to contain the slave oscilloscopes, patient alarm and direct-writing recorder. It was stocked with medication necessary for patient care. One trained registered nurse was assigned for every two patients within the monitoring facility. Convalescent patients with cardiac disease and other patients confined to the ward were cared for by nurses working at the main station.

Patients were admitted to the monitoring facility at the request of their referring physician. Any patient with an acute myocardial infarction, regardless of clinical condition, was accepted. Those patients in whom infarctions were not proven were transferred as soon as possible. Patients without infarctions but with heart disease and rhythm disturbances deemed harmful were also accepted. After discharge from the unit, patients convalescing from infarctions or from major arrhythmias were kept on the same ward as long as possible in order to take advantage of the equipment and nursing experience should serious rhythm disturbances develop later.

Nurses working within the unit were initially given a 30-hour didactic lecture series. In addition, they were instructed in the principles of intravenous therapy and in the taking of standard electrocardiograms. As part of their training, they performed elective cardioversions in the operating room under the supervision of a physician. In anticipation of the responsibility delegated to these nurses, a pay scale for them similar to that of surgical nurses was agreed upon by the hospital administration.

Physicians wishing to admit and to care for patients within the monitoring facility were granted privileges to do so only after instruction in cardiopulmonary resuscitation and in the use of the electrical equipment. In order to assure physician coverage and attendance at any cardiac arrest, a double-call system was arranged. In the event of a resuscitation emergency both the attending physician and the unit director or his appointee were called.

Each patient's electrocardiogram was continuously monitored by precordial electrodes held firmly with paper tape. The monitor was routinely set to alarm at rates above 150 or below 40 with a 7-second preset delay. A photoelectric cell was placed on the ear lobe for qualitative assessment of arterial pressure. This was necessary since occasionally adequate electrocardiograms could not be obtained and deterioration of cardiac function might be evidenced by decline in arterial pressure before electrical deterioration. Whenever possible a nurse was present within the monitoring unit station to view the electrocardiographic traces on the oscilloscope. In this way any trend toward potentially dangerous rhythm disturbances could be noted and the physician notified. On occasion, cardiac arrest was predicted from this viewing of the tracing, and the nurses arrived in the patient's room before the alarm sounded.

The duties and responsibilities of the monitoring unit nurses during cardiac arrest were set forth in written rules of procedure (Table 1). Nurses im-

*Sanborn Division, Hewlett-Packard Co.

TABLE 1.—*Procedure to Follow during Cardiac Arrest in Monitoring Unit*

1. Establish BP, level of consciousness, pulse, nature of rhythm on oscilloscope.
2. *(a) Ventricular tachycardia*
   Massage and ventilate; Lidocaine 100 mg. IV; HCO_3 45 mEq IV; BP support with Metaraminol 100 mg./liter; if shock or unconsciousness, synchronized countershock.
   (b) Ventricular fibrillation
   Immediate DC countershock followed by massage and ventilation; HCO_3 45 mEq IV; Lidocaine and Metaraminol if needed.
   (c) Cardiac asystole or complete heart block with slow ventricular rate
   Massage and ventilation; external pacing if no response; HCO_3 45 mEq IV and isoproterenol 1 ampule (0.2 mg.) or epinephrine 0.5 mg. IV for asystole; isoproterenol 1 mg./liter titrated at 1 cc/min initially for heart block.
   (d) Rapid atrial fibrillation; atrial tachycardia
   Massage and ventilation; HCO_3 45 mEq IV; synchronized shock if condition critical and physician not available.
   (e) PVCs, bursts of ventricular tachycardia
   50-200 mg. bolus IV Lidocaine
3. Nurses are allowed under urgent circumstances to institute pacing or electrical shock, administer medication or to follow prior written directions from the attending physician.

MONITORING UNIT COMMITTEE

mediately begin ventilatory and circulatory support with mouth-to-mouth or bag breathing and closed-chest massage. In cardiac asystole they are authorized to administer intravenously 0.2 mg of isoproterenol or 0.5 mg of epinephrine. The same drugs are titrated intravenously if complete heart block is present. If there is no response, external pacing may be begun. Our experience has led us to believe that no time should be wasted in administering shock to a patient with ventricular fibrillation. Timed drills showed that equipment could be moved into the patient's room, assembled and activated in 45 seconds. Therefore nurses first attempt to defibrillate the patient and then begin resuscitation. Intravenous solutions are running slowly in all patients within the monitoring facility and the nurses are authorized to give intravenous sodium bicarbonate during resuscitative therapy. Lidocaine drips may be manipulated so as to suppress abnormal rhythms. All these measures take place in the absence of a physician, and upon his arrival further therapy is at his discretion.

Results

Information on 241 patients with myocardial infarction monitored for an average of five days is included in Table 2. Fifty had cardiac arrest which, regardless of mechanism, is defined as failure of the circulation — invariably fatal unless adequate resuscitative measures are effected. Twenty-three patients were successfully resuscitated, surviving over 24 hours. Ten of these were discharged ambulatory. Mortality in the hospital for the 241 patients was 16.6 percent whereas for unmonitored patients with myocardial infarction during the same period (Table 3) the mortality was 26.2 percent, with no survivors from cardiac arrest. Review of 240 patients with myocardial infarction seen at this hospital from February 1963 to February 1966 showed an overall hospital mortality of 24.2 percent. This group is mentioned for comparison alone since only limited conclusions can be drawn from these figures and the group does not constitute a valid control.

TABLE 2.—*Cardiac Monitoring Unit (March 1966-August 1967)*

517 patients admitted
50 arrests
23 successful resuscitations
10 survivors
mortality 16.6%

Arrest	*Time*	*Survivors*
9	4 hrs	2
4	12 hrs	—
9	24 hrs	2
4	48 hrs	2
8	72 hrs	3
6	1 week	1
10	over 2 week	—

*Arrhythmias recognized (87.4% of patients)**

	No.
1° block	16
2° block	8
complete heart block	18
atrial prematures	90
nodal rhythm	16
sinus bradycardia	8
atrial tachycardia	13
atrial flutter	25
atrial fibrillation	68
PVCs	174
ventricular tachycardia	32
ventricular fibrillation	23
ventricular standstill	12

*Many patients exhibited multiple arrhythmias.

TABLE 3.—*Unmonitored Patients with Myocardial Infarction (March 1966-August 1967)*

62 patients
16 deaths
mortality 26.2%

Time of death	*No. of patients*	*Arrhythmias defined*	*No. of patients*
1st day	5	atrial fibrillation	3
2nd day	4	atrial tachycardia	1
5th day	2	PVCs	2
1st week	2	ventricular tachycardia	1
		ventricular fibrillation	1

However, a progressive decrease in mortality was noted during each half-year period (Table 4). There was little change in the first six months of operation but a striking improvement was apparent after 18 months. Several factors may have contributed to this increased survival. First, changes in mortality were first apparent after the initial six months of unit operation, the time at which routine standing orders were instituted for emergencies. Second, there was decrease in the number of cardiac arrests. Although successful resuscitation was more frequent, the proportion of survivors did not change appreciably. Therefore prevention may have played as important a role as resuscitation.

Clinical status

Clinical summaries of resuscitated patients are presented in Table 5. The monitored patients who survived seemed to follow a pattern of mild to moderate disease with sudden and catastrophic interference with electrical function and automaticity. All had rhythm disturbances but few appeared to have severe depression of myocardial function. None of them had congestive failure or sustained shock before the onset of rhythm disturbance. In the majority of the surviving patients cardiac arrest had occurred within the first 72 hours of admission. Patients in whom arrest did not occur until a week or more after admission almost invariably died.

Severe shock or congestive heart failure was present in all but five patients who died. The mortality of patients with shock and failure was unimproved when compared with mortality before the monitoring unit was opened. In these patients a variety of primarily ventricular rhythm disturbances was observed before death. Although in a number of cases these disturbances were temporarily reverted by appropriate therapy, survival was not effected. These rhythms therefore were felt to be agonal and indicative of ventricular failure and death.

Avoidable deaths

In four instances, all within the first year of operation, it appeared on review that death might have been prevented. In one patient who died while in the monitoring facility there was inadequate appreciation of the importance of progressive bradycardia, and refractive ventricular standstill occurred. Of the 40 patients who died, nine did so after discharge from the monitoring facility. In three of these cases, summarized below, death appeared preventable.

CASE A.—A 67-year-old woman entered the hospital hypotensive and with electrocardiographic evidence of acute anteroseptal infarct. Congestive failure and atrial fibrillation developed. Digitalis was begun but high grade AV block developed and the drug was discontinued. Because of steady clinical deterioration, a transvenous pacemaker was inserted on the fourth hospital day. After two days with the pacemaker, sinus rhythm was restored. Her condition improved and she was discharged from the monitoring unit after 16 days. Two days later, while sitting in a chair, she suddenly became cyanotic and unconscious. An electrocardiogram showed ventricular fibrillation and resuscitation was unsuccessful.

CASE B.—A 62-year-old man with previous myocardial infarction seven years before, began having increasing chest pain and shortness of breath. An electrocardiogram showed inferior and anterior wall injury. Digitalis was administered. After five days, he was discharged from the unit. Three days later, while he was using the bed pan, cardiac arrest occurred. No electrical activity was evident on electrocardiogram and resuscitation was unsuccessful.

TABLE 4.—*Data on Survival of Patients in Cardiac Monitoring Unit*

Date	*Infarctions*	*Arrests*	*Successful Resuscitation*	*Survivors*	*Deaths*	*Percent*
3-8/66	74	21	5	2	16	21.6
9/66-2/67	81	16	8	3	14	15.9
3-8/67	79	13	10	5	10	12.7

CASE C.—A 60-year-old man with hypertension had onset of crushing anterior chest pain four hours before admission, with electrocardiographic evidence of acute inferior wall infarction. Digitalis was started when atrial tachycardia began. Three days later, there was evidence of a high degree AV block with a ventricular rate of 70. Digitalis was discontinued. Monitoring continued for ten days and the patient was discharged from the unit in sinus rhythm. Following discharge a gallop developed, and then left ventricular failure. Digitalis was again started. On the 16th day in hospital, there was sudden electrical arrest and death.

Superventricular rhythm disturbances

The importance of ventricular rhythm disturbances and of complete heart block has been well established. However, in patients with acute myocardial infarction, ventricular arrhythmia is not the only lethal arrhythmia. Three important atrial rhythm disturbances were noted in this series: Sino-atrial failure, refractive atrial tachycardia, and sinus or nodal bradycardia leading to ventricular standstill or to ventricular escape with ventricular tachycardia.

Sinus node failure is defined electrically as intermittent or permanent loss of sinus discharge with or without nodal capture. The subsequent pacemaker discharge is usually slow and cardiac output inadequate. This arrhythmia occurred in three patients. An unusual example is shown in Figure 1. Conversion of atrial tachycardia resulted in sinus bradycardia and sinus arrest. Hypotension developed and circulatory adequacy was restored only following return to atrial tachycardia. Postmortem examination showed anterior, inferior and atrial infarctions.

Refractory atrial tachycardia led to death in three patients. Although a common disturbance of rhythm, this is an uncommonly severe complication of myocardial infarction. Previous studies have attested the circulatory inadequacy produced by the rapid ventricular rate.[12] For the diseased heart, this represents an exaggerated oxygen need. As shown in Figure 2, the rhythm disturbance may abruptly lead to serious ventricular rhythm abnormalities and death. In these three patients massive intravenous doses of procaine amide, quinidine and lidocaine and frequent electric countershock were unsuccessful.

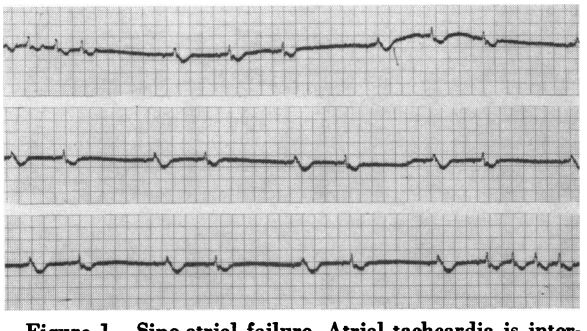

Figure 1.—Sino-atrial failure. Atrial tachcardia is interrupted by sinus arrest and sinus bradycardia. Hypotension ensued and circulatory adequacy returned only during atrial tachycardia.

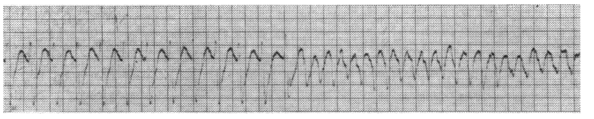

Figure 2.—Sudden transformation of atrial tachycardia with aberrant ventricular response to ventricular fibrillation.

In patients with myocardial necrosis, a critical ventricular rate may be necessary to suppress ventricular escape and ectopy.[9] Reduction in rate may allow reentry beats to drive the heart (Figure 3). Contrarily, slow rates of sinus or nodal origin may terminate in electrical arrest. Such rate-dependent phenomena were found in three patients, one of whom progressed to cardiac arrest and in one of whom unresponsive ventricular tachycardia developed. Knowledge or prediction of such slowing may have prompted appropriate therapy and salvage.

Patients without infarction

Monitoring was requested for a number of patients without myocardial infarction. Summaries

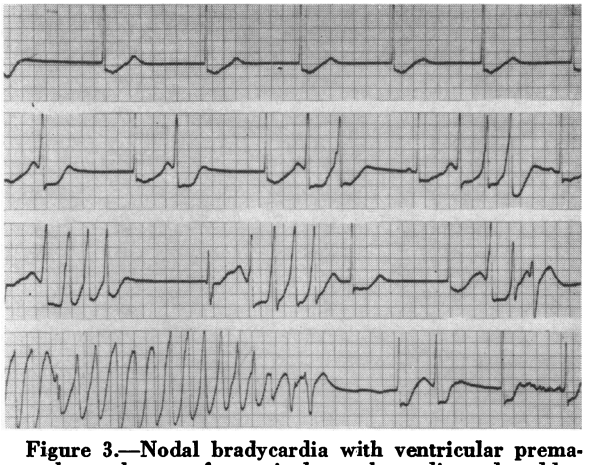

Figure 3.—Nodal bradycardia with ventricular premature beats, bursts of ventricular tachycardia and sudden onset of ventricular fibrillation with spontaneous reversion.

TABLE 5.—*Clinical Data on Resuscitated Patients in Cardiac Monitoring Unit*

				Patients with Myocardial Infarction				
Case	*Age and Sex*	*Pathology*	*Rhythm*	*Shock*	*CHF*	*Therapy*	*Time of Monitor*	*Comments*
1	72 M	Posterior MI	Atrial tachycardia; sinus arrest and bradycardia	Yes	Yes	Shock, lidocaine, HCO_3	5 days	Died; bilateral coronary occlusions; anterior, inferior and atrial infarcts.
2	56 M	Anterolateral MI	Ventricular tachycardia	...	...	Massage, HCO_3, quinidine, lidocaine	8 days	Survived.
3	58 F	Posterior MI	Ventricular fibrillation CHB, PVCs	...	...	Shock, lidocaine, HCO_3, procaine amide, quinidine	10 days	Survived; ventricular fibrillation and infarction following selective injection RCA.
4	68 M	Inferior MI	Intractable atrial tachycardia	...	Yes	Shock, lidocaine, quinidine, digitalis, procaine amide, diphenylhydantoin	11 days	Died 4 days after discharge from unit.
5	74 F	Inferior MI	Ventricular fibrillation	Yes	...	Shock, lidocaine, HCO_3	8 days	Died in renal failure; terminal ventricular fibrillation.
6	67 F	Anterior MI	Atrial fibrillation, CHB; ventricular fibrillation	Yes	Yes	Pacemaker, catheter	16 days	Died in ventricular fibrillation 2 days after discharge from unit; bilateral coronary thrombi.
7	59 M	Anterolateral MI	PVCs, CHB, ventricular fibrillation	Yes	Yes	Shock, lidocaine, HCO_3 digitalis	3 days	Died; 3 prior infarcts; bilateral coronary occlusions.
8	78 M	Anterior MI	Ventricular fibrillation, CHB	Yes	...	Shock, lidocaine	5 days	Died.
9	64 M	LBBB; infarct by enzymes and history	Ventricular tachycardia, fibrillation	...	Yes	Shock, lidocaine	11 days	Died.
10	66 M	Anterior MI	Atrial tachycardia	Yes	...	Shock, metaraminol, quinidine	6 days	Survived; died 9 months later.
11	79 M	Inferior MI	CHB	Yes	Yes	Pacemaker, catheter, digitalis	8 days	Died; refractive failure and unresponsiveness to pacing.
12	42 M	Inferior MI	Ventricular fibrillation	...	Yes	Shock, lidocaine, digitalis	10 days	Died; ventricular fibrillation 6 hours after transfer from unit; intractable failure.
13	62 M	Anterolateral MI	Ventricular standstill	...	...	Isoproterenol	8 days	Survived; subsequent Vineberg procedure.
14	78 F	Inferolateral MI	Ventricular fibrillation	Yes	...	Shock, lidocaine, HCO_3	5 days	Survived.
15	58 M	Inferior MI	CHB	Yes	...	Isoproterenol	6 days	Survived, working.
16	49 M	Anterior MI	Atrial tachycardia; ventricular standstill	Yes	Yes	Shock, procaine amide, isoproterenol	2 days	Died; diabetic; 2 prior infarcts.
17	63 F	Anterolateral MI	Ventricular fibrillation	...	...	Shock, lidocaine, HCO_3	6 days	Survived; working.

18	63	M	Anterior MI	PVCs, ventricular tachycardia	...	...	Procaine amide, shock	5 days	Died 72 hours after discharge from unit; ventricular fibrillation.
19	55	M	Anterior MI	Progressive bradycardia; ventricular standstill	...	Yes	Pacemaker, catheter	10 days	Survived; renal shut down after arrest; now working.
20	81	M	Inferior MI	Ventricular tachycardia	...	Yes	Closed chest massage, HCO_3	7 days	Died 5 days after discharge from unit; intractable CHF; bilateral coronary thrombi.
21	59	F	Inferior MI	Progressive bradycardia; ventricular tachycardia	Yes	...	Pacemaker, isoproterenol, lidocaine	3 days	Died.
22	43	M	Anterior MI	Ventricular tachycardia, CHB	...	...	Shock, pacemaker, catheter	7 days	Survived.
23	54	F	Anterior subendocardial MI	Ventricular tachycardia; ventricular fibrillation	...	...	Shock, HCO_3, lidocaine	7 days	Survived; diabetic.
				Patients without Infarction					
24	77	M	ASHD without infarct	CHB; ventricular fibrillation	Yes	Yes	Pacemaker, catheter, shock quinidine	3 days	Died.
25	44	M	RHD, mitral valve replacement	Ventricular tachycardia	Yes	Yes	Shock, digitalis, quinidine, ethacrynic acid, lidocaine	3 days	Died in intractable CHF.
26	75	F	.....	Paroxysmal ventricular fibrillation; sinus bradycardia	...	...	Shock, lidocaine, ephedrine, digitalis	8 days	Survived; ? digitalis intoxication.
27	43	F	RHD post mitral commissurotomy	Ventricular tachycardia and fibrillation; sinoatrial failure	...	...	Shock	4 days	Survived; digitalis intoxication; now working.
28	53	F	RHD, mitral valve replacement	Ventricular tachycardia and fibrillation; CHB	Yes	...	Shock, quinidine, lidocaine, HCO_3, diphenylhydantoin, pacemaker, catheter	7 days	Survived; digitalis intoxication manifest 3 days after conversion from atrial fibrillation; now working.
29	63	M	ASHD	Ventricular bigeminy and fibrillation	...	Yes	Shock, lidocaine, atropine	8 days	Survived; digitalis intoxication.
30	64	F	Aortic stenosis	Ventricular fibrillation	...	...	Shock, lidocaine, pacemaker, quinidine	2 days	Died; intractable ventricular fibrillation with over 40 individual conversions.

LEGEND

ASHD	arteriosclerotic heart disease
CHB	complete heart block
CHF	congestive heart failure
HCO_3	bicarbonate
LBBB	left bundle branch block
MI	myocardial infarction
PVC	premature ventricular contraction
RCA	right coronary artery
RHD	rheumatic heart disease

of their cases are included in Table 5. These included patients with rheumatic heart disease with atrial fibrillation, patients with digitalis or quinidine intoxication, with paroxysmal rhythm disturbances or complete heart block, and patients with severe aortic stenosis exhibiting Stokes-Adams disease. Cardiac arrest occurred in seven of this group, with successful resuscitation in four. In all four survivors, the supposed mechanism of arrest was ventricular tachycardia or fibrillation due to digitalis intoxication.

Discussion

The results in this group of patients seem to justify the monitoring unit concept, since prevention and therapy of lethal cardiac arrhythmias were large factors in the decrease in mortality. This reduction parallels that found in other series and indicates that a successful program can exist in a community hospital without intern or resident staff where emergency resuscitation is carried out primarily by nurses. Obviously such results represent an understanding and an adaptation to the needs and problems of the community hospital system. The design and organization of such a unit must differ from facilities present in teaching hospitals where full time staff and training physicians are constantly present.

First of all there must be a uniform policy for arrest and resuscitation therapy. When many physicians treat patients, as many manners of care may be found. Analysis of the statistics for the initial six months pointed to a failure of resuscitation in the majority of patients. This failure was attributed to lack of a uniform approach to treatment. After we established a routine policy in cardiac arrest based upon the patient's clinical condition and the arrhythmia present, mortality was substantially improved. Routine orders for cases of arrest enabled nurses to proceed promptly and intelligently during emergencies. This ability is vitally important, since emergency calls may be answered by physicians who ordinarily do not deal with patients with heart disease and who therefore may not be helpful.

Second, to insure a high level of patient care, privileges must be granted to care for patients within the unit. Persons certified to give such care have displayed skill in therapy of patients and familiarity with rhythm disturbances, cardiac drugs, electrical monitoring and therapeutic equipment.

Third, nursing training must not focus only on basic science and interpretation of the electrocardiogram. Rather the emphasis needs to be placed on anticipation and recognition of clinical deterioration, active resuscitation and the use of pacemakers and defibrillators. The training of nurses should not be intended to make them assistants but active participants and therapists in an emergency when a physician is not available. At this point, it may be appropriate to mention that within the last year 13 of 18 successful resuscitations were performed entirely by nursing staff before the arrival of a physician.

The decrease in mortality noted in this and other series quickens interest in how the death rate might be further reduced. If our ten survivors were added to mortality statistics from myocardial infarction, the overall risk of 20.8 percent would not differ greatly from previous results. Monitoring units therefore seem to assure the salvage of viable patients. In patients with shock and congestive heart failure, no appreciable change has been noted since the monitoring unit was organized, and the need for more adequate pharmacologic tools or for assisted circulatory devices is clear.

However, this study indicates several potential ways in which the death rate may be reduced in other potentially viable patients. First, early hospital admission of persons with suspected myocardial infarction is essential. The frequency of lethal ventricular arrhythmia soon after infarction has been stressed.[8] Serious arrhythmia often has been demonstrated either at home or in the hospital within the first few hours after admission.[1,14] In our experience, arrest occurred within the first 24 hours in 23 cases, and four of the patients were revived. Physicians suspecting myocardial infarction may well reduce mortality by prompt action.

Second, patients with infarction appear to fall into three general categories — called here, Groups 1, 2 and 3 — and the category dictates to a great extent the aggressiveness of therapy, the length of monitoring and the possibility of survival. Guided by the clinical appearance of the patient, the nursing staff can predict his course and plot the appropriate therapy necessary.

Group 1 patients are those with sudden catastrophic rhythm disturbances occurring usually within 72 hours after admission. Ventricular function is assumed to be adequate since resuscitation is usually not difficult. None of these patients in the series here reported had persistent shock or

failure and only one of nine died. Patients with rhythm disturbance of this type are few in number.

Group 2 patients have frequent unpredictable changes in rhythm for periods as long as two weeks after infarction. In our series shock and failure were present in patients of this order and were only tenuously controlled by medication. For these patients prolonged monitoring was required. Since the risk of recurrence of arrhythmias was high after discharge, telemetric monitoring for the duration of stay in hospital has been suggested. Arrest in these patients was poorly tolerated and more difficult to reverse and only two have survived.

Group 3 patients have irreversible myocardial damage, shock and congestive heart failure. Rhythm disturbances may be altered but survival is not effected. Therefore emphasis is placed on distinguishing Type 1 and Type 2 patients. The former are discharged from monitoring in four to five days if no significant rhythm disturbances are noted. Patients in Group 2 are monitored for more than one week.

Third, potentially viable patients may be lost in the post-monitoring period. Almost 20 percent of our subjects who died did so after discharge from the unit. As was previously mentioned, three deaths appeared preventable. Consequently it has been our policy to leave convalescent patients with impaired myocardial function or previous arrhythmia on the ward as long as possible to take advantage of equipment and personnel available. All RNs working on the convalescent ward have monitoring unit training and experience. A telemetric system compatible with our monitoring equipment has been purchased.

Finally, our results have suggested that a greater emphasis must be placed on rhythm disturbances previously considered benign. Sino-atrial arrest, atrial tachycardia and sinus and nodal bradycardia may less abruptly and dramatically produce cardiac deterioration, and perhaps it is the fact that they are initially tolerated well which leads to complacency and to a lessened sense of therapeutic urgency. However, suitable treatment should be instituted as quickly as possible, with the same aggressive approach as for ventricular arrhythmias.

There appears to be no reason why monitoring should be restricted only to those with myocardial necrosis. Admission should be governed primarily by need and by the availability of bed space. Our experience with resuscitation of patients with digitalis-induced ventricular tachycardia and fibrillation is impressive since these arrhythmias have not previously responded to electric countershock and are frequently fatal.[11]

It is evident that monitoring units have made an important contribution to immediate survival of patients with rhythm disturbances of many varieties and from many causes. Yet little information is available as to the life expectancy of resuscitated patients and their ability to return to normal life.[4] Our numbers are small and the follow-up is short. However it is significant that of the 14 patients resuscitated, five have returned to work. In follow-ups ranging from one to seventeen months there has been only one death. The patient died of cardiac arrest at home.

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